

as a minimum. It delays operation only a few days and does not increase the operative difficulties.

The author showed that there is a 30 per cent error in diagnosis of the axillary glands in supposedly Group 1 cases. It is probably in this 30 per cent of cases that preoperative radiation has its chance to be of benefit. Let us remember that if we can increase the five-year survival by even five in one hundred, it is just as important to save these five women in Group 1 as in Group 2.

In Group 2 patients, I believe that preoperative irradiation should be used for the same reasons as preoperative irradiation in Group 1. In Group 2, because the surgical results are so poor, a thorough course of radiation should be used rather than a short course. It must be remembered that there are practically no statistics on the value of preoperative protracted fractionated courses. To base our opinions of the value of preoperative radiation on older statistics is like basing an opinion of the value of surgery on late nineteenth century surgery.

Another point on which we disagree is on the value of repeated courses of radiation after surgery. If the cancer was found to be limited to the breast and axilla, we should give one really thorough course of irradiation, such that we would not care to repeat, and rest with that. Most authorities are agreed that the chance of irradiation curing lies in the first attack on the disease and not on repeated attacks.

The number of fields to be used and their size will always be a matter of individual opinion. In general, I use a smaller number of fields and larger total doses per port than the author indicates. From sad experience, I should like to emphasize what the author insinuated, namely, that the ports must be arranged so as not to get large doses of radiation in the lungs, either by crossfire or direct radiation.

DERMATOMYOSITIS*

REPORT OF CASE ASSOCIATED WITH RHEUMATIC HEART DISEASE

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DERMATOMYOSITIS is a rare disease best described as an acute, subacute, or chronic disease of unknown origin, characterized by a gradual onset with vague and indefinite prodromata, and followed by edema, dermatitis, and multiple non-suppurative inflammation of muscle. The first clear-cut cases were described independently by Unverricht,¹ Hepp,² and Wagner³ in 1887.

PATHOLOGY

Early the skin and subcutaneous tissues show edema, infiltration with neutrophils and some areas of hemorrhage. Later there may be desquamation of the surface layers with degeneration of the corium and disappearance of the papillae. The changes in muscle are characteristic. First, there is transudation of fluid and interstitial and perivascular infiltration of leukocytes. The muscle bundles show a patchy involvement, and normal ones may be seen among others showing all stages of degeneration. Later, evidence of regeneration

of muscle appears in scattered areas, and there is much increase in connective tissue.

ETIOLOGY

The cause of the disease is obscure. Certain features suggest an infective origin, namely, the first symptoms frequently following some febrile disturbance such as tonsillitis, influenza, measles, rheumatic fever, etc.; the demonstration often of foci of infection; the febrile course; the presence of splenomegaly in many cases; and the relapsing nature of some cases.

Various organisms have been thought specific by different observers, but in many cases no organisms are found. Probably the disease is a syndrome which may result from a variety of causes. All ages are affected, and the incidence in both sexes is approximately equal. The white races have been almost exclusively affected. Winter seems the commonest time of onset.

SYMPTOMS

Following the vague prodromata, symptoms appear insidiously. These consist of increasing muscular weakness with pain, swelling and rigidity, which is usually widespread and symmetrical, and accompanied by a peculiar exanthem and a brawny edema. The muscles alternately feel firm and tough, or soft and boggy. The limbs are most commonly affected and, in contrast to trichinosis, the muscles of the eyes are not often involved. The rash usually has a peculiar heliotrope color and is distributed over the affected muscles. Later in the disease the rash may resemble various types of skin disease, and as an end-result brownish pigmentation is common. Eventually the skin becomes thickened and may even resemble scleroderma.

A high intermittent or remittent fever may be present during the acute stages, but frequently is of mild character. The spleen is often enlarged. Leukocytosis is common but, typically, no eosinophilia is present. Creatinuria has been reported. This is not primary, but is undoubtedly secondary to the anatomical changes in the muscles.

Death is common during the acute stage, usually from intercurrent infection. If the patient lives long enough, the pain and edema begin to subside and fibrosis of the skin and muscles develops, resulting in marked deformity. The skin then becomes hard and inelastic and bound down to the underlying muscles, which may be felt as board-like wasted bands.

Recovery is often accompanied by residual weakness and contractures, and certain areas of skin may always remain sclerotic. However, it is possible for the patient to recover completely after a most severe attack. Unfortunately, relapses are common. About 50 per cent of the reported cases die, some in the acute stages, but more after months of illness.

DIFFERENTIAL DIAGNOSIS

Dermatomyositis, during its acute stage, may so simulate trichinosis that early reports used the title "Pseudo-trichiniasis." However, in trichinosis, changes are usually most prominent in the

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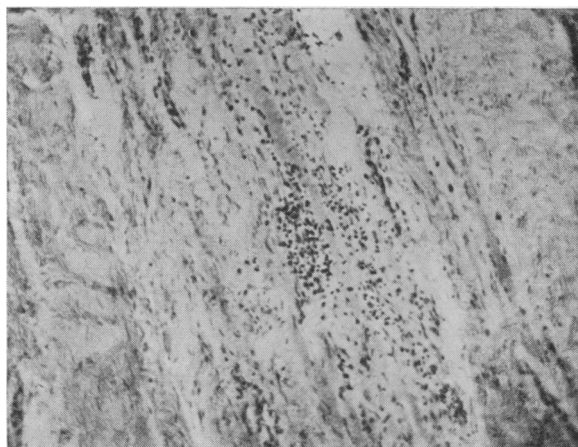


Fig. 1

Fig. 1.—Photomicrograph of muscle from calf. Note infiltration of leucocytes about muscle bundles and increase in connective tissue. (X105)

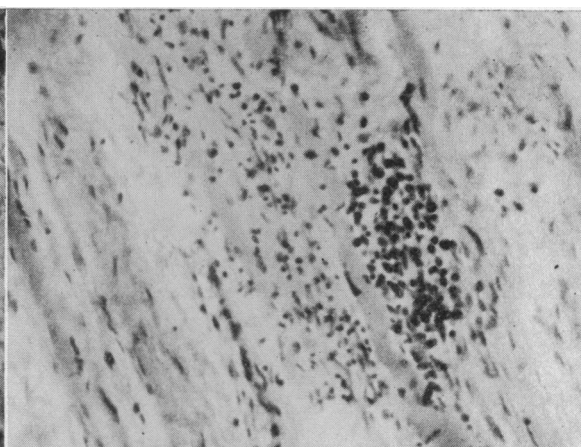


Fig. 2

Fig. 2.—Photomicrograph of muscle from calf. Varying degrees of degeneration of muscle are shown. Note wavy contour of fibers and fibrosis. (X210)

ocular muscles. Eosinophilia, which is uncommon in dermatomyositis, is always present in trichinosis, and larvae may be found in the blood or muscle.

Polyneuritis may cause confusion, especially as in some cases of dermatomyositis the nerves are involved as well. However, in uncomplicated polyneuritis, edema and muscular swelling are absent.

Scleroderma can be excluded in the chronic cases only by the presence of myositis. Many believe that the two diseases are different manifestations of the same morbid process, for sclerodermatous changes often accompany dermatomyositis.

TREATMENT

All forms of therapy have been advocated, none of which have been specific. Stress should be laid upon the prevention of deformity and upon various orthopedic and physical therapeutic measures which should be employed if the patient recovers. The patient presented was treated with autogenous vaccine, as was one other patient reported by McGarrah.⁴ Ordinarily no bacteria are found in biopsy specimens. McGarrah isolated a culture of *Staphylococcus albus* from a nasal smear. As this gave a positive intradermal reaction, a vaccine was made and administered with apparent benefit.

REPORT OF CASE

M. B., a 22-year-old female milliner, of Italian descent, was first seen on the Medical Service of the University of California Hospital in September, 1935, complaining of pigmentation and muscular weakness of two years' duration. Eight years previously, at the age of fourteen, she was vaccinated for smallpox. A small brown area of pigmentation appeared at the site of vaccination which extended over the entire upper left arm during the next few years, and similar areas of pigmentation appeared on the right arm. This was accompanied by a decrease in size of the left arm. Two years before entry, at the age of twenty, she noted nodules in the calf of the left leg and weakness of both legs. This was followed by edema and she was treated for congestive cardiac failure, with improvement. However, contractures of her calf muscles developed, so that she was unable to walk flat-footed. For eighteen months before entry she was confined to bed with generalized muscular weakness and stiffness. With the onset of stiffness the pigmentation previously noted spread over the entire body. During the last month of her acute illness she had had fever and profuse perspiration, and had lost

thirty-five pounds in weight. No muscular pain had been noted. Infected ingrown nails of both great toes had been present about three years. Her past history was not remarkable, except for "growing pains" at thirteen and fourteen years of age.

Examination in 1935 revealed an emaciated girl with fixation of elbows, knees, and hips in 90 degrees flexion. The ankles were fixed in plantar flexion. Generalized pigmentation was present, with the exception of the nose, palms, and soles of the feet. The pigmentation was mottled and of a dirty-brown color. The pigmented area about the left upper arm had sharply demarcated borders. All extremities were wasted, and all muscles felt indurated and cord-like to palpation. Slight generalized pitting edema was present. The mouth could be only partially opened. The heart was slightly enlarged to the left and right. The rhythm was regular. A presystolic murmur, ending in a booming first sound and followed by a blowing systolic murmur, was heard at the mitral area. The pulmonic second sound was accentuated and reduplicated. The blood pressure was 116 systolic, and 70 diastolic. The abdomen showed an enlarged spleen at the left costal margin. Clubbing of the fingers was present. The left great toe showed an infected ingrown nail. The patellar and abdominal reflexes were barely elicited.

Extensive laboratory procedures, including routine urine and stool examinations, Wassermann test on the blood, phosphorus and calcium determinations of the blood, culture of the blood, gastric analysis and spinal fluid examination, were normal. The blood showed a mild secondary anemia and leukopenia. No eosinophilia was present. Creatinuria was present, the twenty-four-hour output of creatin being 98 milligrams and of creatinin being 218 milligrams. Culture of the toe showed *Staphylococcus albus* and *Bacillus subtilis*. A biopsy from the right calf showed characteristic lesions of dermatomyositis (Figures 1 and 2). Culture of tissue obtained at biopsy showed nonhemolytic streptococci.

X-ray films of the chest showed the heart to be enlarged and of a mitral configuration. Films of the lumbosacral spine showed extreme lumbar lordosis. Films of the elbows showed no lesions of bone or joint.

While under observation the patient was febrile, the fever being remittent at times and intermittent at others, reaching maximum of 40 degrees Centigrade. A vaccine was prepared from a mixture of streptococci isolated from a stool culture, and the muscle biopsy and staphylococci from the infected toe. This vaccine was administered intravenously at weekly intervals. With large doses temporary exacerbations occurred, but with small doses improvement appeared. Following the subsidence of the acute involvement, exercises and physical therapy were instituted. At this time the patient was unable to feed herself or to turn in bed. During the past two years improvement has gradually occurred, until now she is able to walk alone with difficulty and can care for herself in bed.

COMMENT

The history and findings of the patient reported are typical of dermatomyositis and show the diagnostic triad of eruption, edema and myositis. Unusual features of this case were the absence of pain, the presence of mitral stenosis, and the isolation of organisms in the biopsy specimen. The onset in this patient was very gradual, and an organism of low virulence, undoubtedly introduced at the time of the vaccination, slowly spread throughout the body over the next few years.

Dermatomyositis may at times involve the myocardium, but never causes valvular lesions as seen in rheumatic heart disease. The mitral stenosis was probably due to rheumatic infection, as manifested by the "growing pains." These preceded the vaccination and were probably independent of the dermatomyositis.

Apparently, an autogenous vaccine was of value in this patient as in that of McGarrahan. Unfortunately, it is seldom that organisms can be isolated from muscle, and it is this fact that has cast doubt on the infectious etiology of the disease. It is of utmost importance, however, that contractures be prevented as much as possible, and that fixation of limbs occur in positions favorable to physiologic function. Following the subsidence of the acute disease, proper massage and graded exercises and manipulation are of inestimable value in rehabilitation.

IN CONCLUSION

1. A case of dermatomyositis is reported which followed vaccination for smallpox and which was associated with rheumatic heart disease.

2. Because of its apparent benefit in this case, further trial of autogenous vaccine in the treatment of dermatomyositis is indicated.

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REFERENCES

1. Unverricht, H.: Polymyositis acuta progresiva, *Ztschr. f. klin. Med.*, 12:533-549, 1887.
2. Hepp, P.: Über Pseudotrichinose, eine besondere Form von acuter parenchymatöser Polymyositis, *Berl. klin. Wchnschr.*, 24:297-299, 1887.
3. Wagner, E.: Ein Fall von acuter Polymyositis, *Deutsch. Arch. f. klin. Med.*, 40:241-266, 1887.
4. McGarrahan, J. D.: Dermatomyositis, *J. A. M. A.* 102:680-681 (March 3), 1934.

DISCUSSION

ROBERT W. LANGLEY, M.D. (1930 Wilshire Boulevard, Los Angeles).—The condition of dermatomyositis occurs infrequently. Its recognition justifies attention and recording. The outlook is usually quite poor. There is little a cardiologist may contribute to this subject. Doctor Kellogg states that dermatomyositis may at times involve the myocardium, but I have been unable to substantiate this statement from any proved cases in the literature. Two fatal cases were recently reported by members of the staff of the Hospital of the Good Samaritan in Los Angeles. Autopsies on both cases failed to show myocardial damage, either gross or microscopic.

There is no question about the diagnosis of rheumatic heart disease of moderately severe degree in this case. The congestive heart failure responded satisfactorily to medical treatment.

Doctor Kellogg suggests, quite rightly I think, that rheumatic infection was associated with, but independent of the dermatomyositis. There may be some relationship between scleroderma and dermatomyositis. Cases have been

reported where a transition appears to have taken place. This writer has observed a case of scleroderma associated with rheumatic heart disease. No relationship between the two seemed apparent.

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HIRAM E. MILLER, M.D. (384 Post Street, San Francisco).—Dermatomyositis is a comparatively rare disease, and it is always of value to report the findings in such unusual cases as this one. The prodromal symptoms in this woman were of very slow onset, extending over a period of eight years or more. She had marked contractures of her leg muscles; pigmentation gradually covered her entire body, but there was no history of an actual dermatitis. She had an associated valvular heart disease, with clubbing of the fingers. Streptococci were obtained by culture from a biopsy taken from the leg muscles. There are probably a number of diverse and only remotely related conditions classified under the symptom-complex of dermatomyositis.

I have observed several patients with this disease. They all have had a dermatitis on the upper face, eyelids and, in most instances, on the extremities. Many of them died due to paralysis of the visceral muscles generally of the respiratory tract. The dermatitis in most instances closely resembled disseminated lupus erythematosus. An associated leukopenia is also observed in this disease. In my experience, a leukopenia is generally found as is recorded in this case, and not a leukocytosis as is stated in the paragraph on symptoms.

I have seen various types of treatment used in this disease, but have not been convinced that any of them have materially changed the course of the disease. Some of the patients recovered and some of them died.

I do not believe that vaccination has played any part in causing the disease in this patient. I think it is unfortunate that this phase of the condition has been given such a prominent place in the conclusions.

INSULIN SHOCK THERAPY IN DEMENTIA PRAECOX: A REPORT OF A SERIES OF CASES*

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INTRODUCTION.—The treatment of dementia praecox by hypoglycemic shock, according to the method of Sakel, is largely empirical in character. The determination of its value can only be made by clinical application to a large number of cases over a period long enough to see the proportion of successes and failures. As the last four years have furnished much data, it is well for us to review the results and try to ascertain if this dramatic form of therapy merits a place in psychiatric practice.

EUROPEAN REPORTS

The reports from European clinics are much larger than from those in this country. Recent literature indicates that about two thousand patients have been treated all over the world. The greater magnitude of the work abroad may be due to its earlier use there; but this also leads one to believe that it has been given wider application than in America. Sakel¹ first reported 104 cases completed in 1937, in which series there were 70.7 per cent

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